Amendment to the Specification

Please replace the paragraph beginning at page 10, line 3, with the following amended paragraph:

FIGs. 2A and 2B are a pair of graphs showing that HSV amplicon-delivered Aβ antigens elicit marked humoral responses. Helper virus-free HSV amplicons ($1x10^5$ transduction units) were delivered subcutaneously to APP_{Swe}-overexpressing transgenic mice (Tg2576) at six weeks of age. Serum was obtained from each vaccinated mouse according to the schema illustrated in FIG. 1B, and 1:256 dilutions were analyzed by ELISA. Levels of antigen-specific antibodies arising from each vaccination were corrected using serum isolated from HSVlac control mice, and are expressed as "Corrected Absorbance @ 450 nm" for a subset of timepoints. ~~FIG. 1A~~ FIG. 2A is an ~~a series of photomicrographs demonstrating that~~ analysis of sera isolated from vaccinated mice by α-Aβ ELISA showing ~~showed~~ that both amplicon-expressed Aβ immunogens were capable of eliciting Aβ-specific humoral responses. Responses induced by the Aβ/TtxFC immunogen elevated at most assay time points and were more durable than those elicited by HSVAβ. FIG. 2B is a graph showing the ~~that~~ analysis of α-TtxFC antibody titers by ELISA; TtxFC responses were specifically generated only in HSVAβ/TtxFC-vaccinated mice. Error bars represent standard deviation, while "*" indicates statistical significance ($P < 0.05$) between HSVAβ/TtxFC and HSVAβ values at same timepoint.

Please replace the paragraph beginning at page 10, line 19, with the following amended paragraph:

~~FIGs. 3A-3F are graphs~~ FIG. 3 is a graph showing that the antibodies elicited by HSV amplicon-delivered Aβ/TtxFC are more Th2-like and more mature than those elicited by HSVAβ. Isotypes of α-Aβ antibodies were determined by ELISA using sera obtained from vaccinated Tg2576 mice at the 4-month post-treatment timepoint. Levels of Aβ-specific antibody isotypes arising from each vaccination were corrected using serum isolated from HSVlac control mice, and are expressed as "Corrected Absorbance @ 450 nm". Error bars represent standard deviation. Marked differences in isotypes were observed between animals

receiving the two Aβ immunogen forms. HSVAβ-treated mice harbored exclusively α-Aβ antibodies of the IgM class while the HSVAβ/TtxFC-immunized Tg2576 mice produced antibodies primarily of the IgG1 isotype, with detectable levels of the IgA class. In addition, there existed κ light chain bias in the α-Aβ antibody pool obtained from HSVAβ/TtxFC-injected mice.

Please replace the paragraph beginning at page 11, line 1, with the following amended paragraph:

~~FIG. 4 is a graph~~ FIGs. 4A-4F are graphs showing that HSVAβ-vaccinated mice exhibit enhanced pro-inflammatory molecule expression profiles in the hippocampus, as assessed by quantitative real-time RT-PCR. Tg2576 and non-transgenic littermates received equal numbers of virions ($1x10^5$ transducing units) subcutaneously at 8 and 12 weeks of age and animals were sacrificed one week after the final injection. Total RNA was isolated from microdissected hippocampus from one hemisphere of each mouse (n=4 per group). Levels of various pro-inflammatory molecule transcripts were determined using quantitative "real-time" RT-PCR, and values expressed as relative transcript level (mean ± standard deviation) normalized to levels of a GAPDH internal control target. Injection of Tg2576 mice with HSVAβ led to a specific up-regulation of IFN-β (A), IFN-γ (B), IL-6 (C), MIP-2 (D), and TNF-α (E) transcripts as compared to HSVlac-vaccinated Tg2576 mice. HSVAβ-treated non-transgenic mice did not exhibit these enhanced pro-inflammatory transcript profiles. Assessment of TNF-β (F) expression determined a positive trend in HSVAβ-vaccinated Tg2576 mice but the difference as compared to the HSVlac-treated cohort did not reach significance. Similar analyses of HSVAβ/TtxFC-treated mice of either genotype showed only a statistically significant up-regulation of the chemokine MIP-2, while all other markers in the hippocampus of these animals remained similar to HSVlac controls. Error bars represent standard deviation, while "*" indicates statistical significance ($P < 0.05$) between HSVAβ or HSVAβ/TtxFC and HSVlac control values.

Please replace the paragraph beginning at page 11, line 20, with the following amended paragraph:

FIGs. 5A and 5B ~~are a pair of graphs showing~~ show that HSVAβ/TtxFC-treated Tg2576 mice exhibit altered plaque morphology and reduced numbers of small Aβ-immunopositive deposits. To qualitatively and quantitatively assess brain-harbored Aβ burden, Tg2576 mice and non-transgenic littermate controls (Non-Tg) receiving HSVlac (n=3) or HSVAβ/TtxFC (n=4) were sacrificed at 11 months of age, perfused, and brains processed for immunocytochemical analysis. FIG. 5A ~~is a graph of~~ is a series of photomicrographs showing representative immunocytochemical staining with the α-Aβ antibody 6E10, of brain sections ~~highlighted~~ highlighting marked differences in the appearance of Aβ deposits between HSVlac- and HSVAβ/TtxFC-vaccinated Tg2576 mice. Background staining in Non-Tg mice is also shown for comparison purposes. Brain-harbored Aβ deposits appeared qualitatively different in HSVlac-treated Tg2576 mice than in HSVAβ/TtxFC-immunized counterparts. FIG. 5B is a graph showing quantitative morphometric analyses performed to enumerate differences in brain Aβ plaque burden in 11 month-old Tg2576 mice. The numbers of 6E10-immunopositive deposits were determined for each of three deposit area ranges (50 μm^2 to 200 μm^2, 200 μm^2 to 500 μm^2, and deposit areas > 500 μm^2). HSVAβ/TtxFC vaccination resulted in a decrement in numbers of deposits occupying the smallest area. Error bars represent standard deviation, while "*" indicates statistical significance ($P < 0.05$) between HSVAβ/TtxFC and HSVlac values in same range of deposit size.

Please replace the paragraph beginning at page 23, line 25, with the following amended paragraph (underlining of journal volumes in original):

By "essential HSV genes", it is intended that the one or more vectors include all genes that encode polypeptides that are necessary for replication of the amplicon vector and structural assembly of the amplicon particles. Thus, in the absence of such genes, the amplicon vector is not properly replicated and packaged within a capsid to form an amplicon particle capable of adsorption. Such "essential HSV genes" have previously been reported in review articles

by Roizrnan (*Proc. Natl. Acad. Sci. USA* 11:307-l 13, 1996; *Acta Viroloeica* 43:75-80, 1999). Another source for identifying such essential genes is available at the Internet site operated by the Los Alamos National Laboratory, Bioscience Division, which reports the entire HSV-1 genome and includes a table identifying the essential HSV-1 genes. ~~The genes currently identified as essential are listed in FIG. 3.~~